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Short Communication

Posterior Reversible Encephalopathy Syndrome after Lumbar Puncture

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Abstract

Traditionally, Posterior Reversible Encephalopathy Syndrome (PRES) is associated with hypertensive crisis, preeclampsia, and treatment with immunosuppressive agents, sepsis, and either renal or hepatic failure. Less common etiologies include intracranial hypotension in the setting of dural tear from spine surgery [1], epidural anesthesia [2] and Lumbar Puncture (LP) [3]. We report a case of PRES in a patient with Trigeminal Neuralgia (TN) in the context of large volume LP in a patient with trigeminal neuralgia.

Keywords: Lumbar puncture; PRES; Visual loss; Trigeminal neuralgia

Introduction

A 66 year old woman with treatment resistant to carbamazepine, oxcarbazepine, gabapentin, and corticosteroids Trigeminal Neuralgia (TN) presented to the emergency department with a worsening headache after a lumbar puncture. TN was resistant to carbamazepine, oxcarbazepine, gabapentin, and cortico-steroids. MRI of the brain revealed a small encephalocele in the right petrous bone and empty sella tursica. Idiopathic intracranial hypertension was suspected as a potential contributing factor to her symptoms and diagnostic Lumbar Puncture (LP) was performed in the outpatient intervention radiology setting. Shortly after she developed a progressively worsening headache along with worsening of TN attacks and was admitted to the hospital for further management. On the third day of her hospitalization patient reported problems with the vision along with worsening of the headache localized to the occipital regions and now with strong positional component. She has become progressively more confused and was found to be blind in both eyes. The exam was consistent with cortical blindness. Systolic BP was below 130 mm Hg. The symptoms of headache, confusion and visual loss could be attributed to stroke in the posterior circulation, arterial dissection, occipital lobe seizures, PRES or Giant Cell Artheritis.

Investigations

EEG demonstrated no seizures. Blood inflammatory markers were unremarkable. CBC and CMP were within acceptable limits. Repeated MRI of the brain demonstrated characteristic Fluid-Attenuated Inversion-Recovery (FLAIR) hyperintensities in the posterior cerebral regions consistent with PRES, accompanied by the lepto-meningial enhancement (Figure 1).

Treatment and Outcome

Patient was treated with non-invasive supportive measures. Epidural blood patch was considered, but was not performed due to continuous clinical improvement. TN was managed with Lamotrigine. The headache improved and vision gradually returned to baseline over the next few days. TN attacks subsided.

Discussion

Presumed pathophysiology of PRES is loss of cerebro-vascular autoregulation and endothelial dysfunction leading to increased capillary permeability resulting in vasogenic edema in the brain [4]. The posterior circulation is more vulnerable because of the unequal distribution of autonomic innervation, with less innervation of the vessels in the posterior cerebral circulation rendering them most vulnerable to the effects of hypertension and hyperperfusion [5], resulting in bilateral, and overall symmetric vasogenic edema in the subcortical and cortical parieto-occipital regions [6]. Association of immunomodulatory agents and PRES is somewhat less clear but has been linked to blood brain barrier disruption secondary to endothelial dysfunction through cytotoxic or cytokine mechanisms [7].

Clinical presentation includes headache, seizures, visual disturbance, dizziness, and mental status changes [8]. MRI is a diagnostic test of choice. Vasogenic edema is visualized on the FLAIR sequence. Cytotoxic edema is assessed on Diffusion-Weighted (DWI) and correlated with Apparent Diffusion Coefficient (ADC) images. The presence of hemorrhage, if any, is best appreciated on Gradient Echo (GRE) sequence [6].

In our case we postulate that a rapid reduction in CSF volume lead to intracranial hypotension and precipitated the onset of PRES. As per Monro-Kellie Doctrine, with the intact skull the sum of the volumes of brain parenchyma, CSF and blood is constant. In reverse, a rapid

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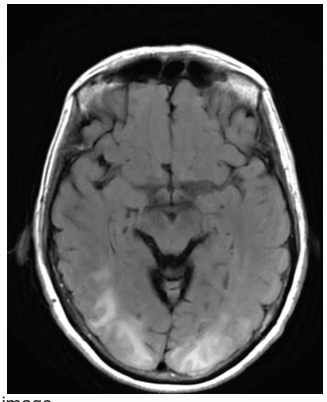

Figure 1: Axial T2 FLAIR image.

reduction in CSF volume is compensated by the increase in blood volume with venous vasodilatation resulting in vasogenic edema. The meningeal enhancement from dilated venous sinuses and engorged capillaries are the likely explanation of contrast enhancement.

Treatment of PRES involves reversal of the underlying mechanisms and removal of offending agents. In hypertensive emergencies, normalization and maintenance of blood pressure is the mainstay of treatment. Discontinuation of immunesuppressive agents is needed as appropriate [7,8]. If a CSF leak is suspected either from incidental or iatrogenic dural rupture, an autologous epidural blood patch can be placed to both stop the CSF leak and increase CSF pressure needs [9].

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